EXHIBIT 1

Expert Report of Walter N. Kernan, M.D.
July 1, 2010

I have personal knowledge of the following statements and opinions, which are true and accurate to the best of my knowledge and information. I am competent and qualified to express the statements and opinions contained in this document.

1. Background and Qualifications

1.1 I am a board-certified general internist with special training and expertise in clinical epidemiology. After completing three years of residency training in internal medicine, I completed a two year fellowship in clinical epidemiology, which is a field of science that applies epidemiologic and other research methods to the study of health problems that affect individual patients treated by practicing physicians. Problems of prognosis, risk or causation, diagnosis and treatment are all within this field. After fellowship in 1989, I joined the faculty at Yale School of Medicine where I am a professor of internal medicine.

1.2 My career at Yale School of Medicine is focused on the care of adult patients, including the prevention and management of chronic disease. I am a member of the Section of General Internal Medicine within the Department of Internal Medicine. I teach general medicine to residents and students, and direct a required clerkship for third-years students during which they acquire core competencies in office-based internal medicine. I follow a panel of adult patients as their primary care provider and assist in the care of over 5,000 adults who attend the Primary Care Center at Yale New Haven Hospital. Many of our patients receive prescriptions for digitalis preparations.

1.3 Over the past 16 years, my research has been focused on medical education, chronic disease epidemiology, and improving the care of patients with stroke. In 1985, I published a prospective observational cohort study on the incidence and risk factors for digitalis toxicity among elderly persons. Using data from a large population-based cohort in New Haven, Connecticut, my colleagues and I demonstrated that the incidence of hospitalization caused by digitalis toxicity was approximately 4.2% over 6 years[1]. Risk factors for toxicity included co administration of quinidine therapy. I am currently the principal investigator on a clinical trial that is funded by the National Institutes of Health.

1.4 My experience, training, and research expertise qualifies me to offer opinions regarding matters related to use of digitalis in medical practice and, specifically, digitalis toxicity. A copy of my curriculum vitae is attached as exhibit A.

2. Materials Reviewed and Relied Upon

2.1 Listed below are documents I have reviewed to date with respect to the opinions contained in this report. My opinions, however, are substantially based on a broader body of scientific literature that I have read over many years. My opinions are also based on my training, experience, and research expertise.

2.2 Various Legal Documents
Defendant's Position Statement – in re: Digitek®, Products Liability Litigation, MDL No. 1968

2.3 FDA Documents
"Facts & Myths about Generic Drugs."
http://fda.gov/Drugs/ResourcesForYou/Consumers/BuyingUsingMedicineSafely/Und. . .. Accessed 9/9/09.

2.4 Company Records
FDA-approved Digitek Package Insert (revised November 2000)

2.5 Journal Articles Cited in exhibit B.

3. Prior Testimony

3.1 Attached as exhibit C is a list of all other cases in which I have testified as an expert witness at trial or in deposition in the last four years.

4. Compensation

4.1 I will be compensated $400.00 per hour for review of case material, $450.00 per hour for deposition testimony, and $4500.00 per day or part thereof for court testimony.

5. Brief Summary of Clinical Pharmacology and Toxicity of Digoxin

5.1 Clinical Uses

5.1.1 Digoxin is the most commonly prescribed member of a class of medications called cardiac glycosides. One of the oldest medications in current use, digoxin is approved by the US Food and Drug Administration for the treatment of heart failure and atrial fibrillation. Heart failure is a common condition in which the pump action of the heart fails to the point that tissues in the body, including the muscles and kidneys, do not receive enough blood flow. Advanced heart failure leads to shortness of breath as fluid backs up into the lungs, swelling of the legs, exercise intolerance, fatigue, and a variety of other problems. There are several causes for heart failure; digoxin is most effective in

controlling symptoms when the cause is systolic dysfunction (essentially a weak heart muscle that does not pump well). In this situation, digoxin improves the pump action of the heart. Digoxin has been shown to improve exercise tolerance and reduce risk for decompensation requiring hospitalization. It probably does not reduce all-cause mortality but in selected persons it may reduce risk for death from heart failure[2-4].

5.1.2 Atrial fibrillation is a condition in which the normal, regular, electrical origin of the heart beat in the atria is replaced by chaotic, irregular electrical activity in the atria that typically results in a rapid heartbeat. Digoxin slows the heart rate by interfering with the conduction of the electrical impulse from the rapidly beating atrial to the ventricles. It is most commonly recommended for use to control the heart rate among patient with atrial fibrillation who also have heart failure, left ventricular dysfunction, or a sedentary lifestyle[5].

5.2 Absorption, Distribution, Metabolism, and Excretion

5.2.1 To understand the potential toxicity of oral digoxin, it is important to understand how it travels through the body after oral ingestion. In general, 60% to 80% of an oral tablet dose is absorbed[6]. The rest is excreted unchanged in the feces or metabolized to inactive compounds by gut flora. The peak serum concentration is reached in 1 to 3 hours. After absorption from the gut, digoxin is distributed widely in tissues of the body so that the apparent volume of distribution is large (4-7 l/Kg). The volume of distribution is the "fluid volume that would be required to contain the total amount of absorbed drug in the body at a uniform concentration equivalent to that in the plasma at steady state."[7]. Therapeutic serum levels range from 0.8 to 2.0 ng/ml, but some data suggests that even lower concentrations may still be associated with benefit in persons with heart failure. Levels less than 2.0 ng/ml are not typically associated with toxicity. In the treatment of atrial fibrillation, physicians commonly adjust the dose of digoxin to bring the heart rate into the normal range (i.e., 60-100 beats/minute); serum level exceeding 2.0 ng/ml may be accepted as long as there are no symptoms of toxicity.

5.2.2 A small portion of absorbed digoxin is metabolized in the liver, but most of the drug is excreted unchanged in the urine. The elimination half life (time until the serum concentration falls by 50%) is 1.5 to 2 days in persons with normal renal function.

5.3 Dosage

5.3.1 Digoxin is available for oral and intravenous administration. The intravenous preparation is now used only rarely for the emergency treatment of atrial fibrillation. It has been replaced by other agents, particularly beta blockers and calcium channel blockers, which have a more rapid onset of action. The intravenous dose is used even less often for heart failure. When administered intravenously, digoxin is typically given at an initial dose of .125 or .25 mg. It may be repeated every two hours up to a maximum dose of 1.5 mg.

5.3.2 For oral use, digoxin is available as an oral elixir (0.05 mg/mL), tablets (0.125 mg or 0.25 mg), and capsules (0.1 mg and 0.2 mg). Individual response to digoxin is highly variable and daily oral adult doses typically vary from 0.125 mg to 0.5 mg.

5.3.3 The dosage of digoxin is adjusted for conditions which can affect renal clearance, particularly age and known renal impairment. In renal impairment, the required daily dose may be less than .125 mg.

5.3.4 Digoxin may interact with other drugs. The most common and potentially important interactions are with drugs that are likely to increase serum drug concentrations of digoxin (e.g., quinidine, verapamil, amiodarone, clarithromycin) and with diuretics that can reduce serum potassium[4, 6, 8].

5.3.5 Physicians and other clinicians who prescribe digoxin for their patients must monitor for therapeutic response and toxicity. During routine follow-up, physicians should ask about symptoms of heart disease and examine patients for signs of heart failure or rapid heart rate. They should also ask about symptoms of possible toxicity, such as nausea, loss of appetite, diarrhea, or visual change. Once a stable digoxin concentration has been obtained, therapeutic drug monitoring is not necessary for most patients with heart failure and normal renal function who are taking 0.125 mg/ day because the serum drug concentration can be reasonably predicted from the dose[4, 9]. Therapeutic drug monitoring is primarily indicated in suspected toxicity, compliance investigation (when indicated), therapeutic failure, and in the presence of events that may alter digoxin serum drug concentration[4]. Events that may cause the digoxin serum concentration to rise include addition or subtraction of medications that affect potassium or digoxin metabolism, altered renal function, dehydration, and improper medication administration.

5.4 Toxicity

5.4.1 Digoxin therapy is associated with important dose-related toxicities primarily in two body systems: gastrointestinal and cardiac[8, 10, 11].

5.4.2 Gastrointestinal toxicities

5.4.2.1 The common gastrointestinal toxicities include loss of appetite, diarrhea, nausea, and vomiting. These are non-specific symptoms which may have many other causes.

5.4.3 Cardiac Toxicity

5.4.3.1 Digitalis toxicity may result in a variety of rhythm disturbances[8, 12]. Most commonly, these include atrial and ventricular ectopy, junctional tachycardia, AV block, and the combination of AV block and accelerated rhythms. These disturbances, however, are non-specific and may occur in the absence of digoxin therapy. In a large recent placebo-controlled trial of digoxin therapy, the risk of suspected digoxin toxicity during an average of three years was 11.9% among patients assigned to digoxin and 7.9% among

those assigned to placebo; the most common reason for suspected toxicity was an arrhythmia[3].

5.4.4 Other manifestations of digoxin toxicity

5.4.4.1 Digoxin therapy has been associated with visual disturbance, particularly blurry vision or distorted color vision. Other reported reactions include allergy, confusion, apathy, hyperkalemia, headache, fatigue, and dizziness have been described. With the possible exception of distorted color vision, most of the symptoms of digoxin toxicity are non-specific and may have many other causes.

5.4.5 Risk factors for digoxin toxicity

5.4.5.1 With the exception of hypersensitivity reactions, risk for digoxin toxicity is directly related to serum level. Factors which tend to increase serum levels may increase risk for toxicity. Examples include older age (which is associated with reduced renal function and reduced lean body mass), chronic kidney disease, and acute illness that results in reduced renal function. Recently, increased risk for toxicity has been associated with the transition between the hospital and outpatient care, a time when patients are often ill and hand-off errors may occur [13]. Some drugs, such as quinidine, amiodarone, verapamil, clarithromycin may affect digoxin absorption, distribution, metabolism, or elimination and increase serum levels[14-16].

5.4.5.2 Despite the fact that risk for digoxin toxicity is directly related to serum levels, there is marked inter-individual variability in tolerance of similar serum levels. Some of this relates to specific factors which can affect the cellular response to digoxin. Factors that tend to increase risk for toxicity include hypokalemia (low potassium in the blood), hypomagnesemia (low magnesium in the blood), hypoxemia (low oxygen in the blood), and acute coronary ischemia[9]. Drugs that deplete serum potassium, such as thiazide diuretics, may increase risk for cardiac toxicity.

6. Opinions

6.1 I have been asked for my opinions regarding the assessment of risks and benefits for individual patients or group of patients who are prescribed digoxin.

6.2 Evaluating the potential causal association between digoxin and a clinical event can be improved with the use of simple classification schemes.

6.2.1 Clinicians are often challenged to judge if a particular patient's symptom is caused by a drug he or she is taking. To help in making judgments about potential causal associations, several sets of criteria have been proposed. Most include some or all components of the criteria proposed by Austin Bradford Hill in 1965: strength, consistency, specificity, temporality, biologic gradient, plausibility, coherence, experimental evidence, and analogy[17]. Of these, temporality is a necessary condition of

causation (i.e., the exposure must occur before the disease). Even these well-known criteria (as acknowledged by Austin Hill himself), however, are regarded as imperfect. Causation cannot usually be established with certainty by application of these or other available criteria. A common error in clinical care is to draw inappropriately definitive conclusions during application of causal criteria.

6.2.2 To improve classification of causation in potential adverse drug reactions, Michael Kramer and colleagues developed a clinical instrument (an algorithm) that considered 6 axes of information[18]:

I. Previous general experience with the drug.
- Has the observed clinical manifestation (potential adverse reaction) been observed previously in association with the drug?

II. Alternative etiologic candidates
- Is there an alternate explanation for the clinical manifestation and can the clinical manifestation be a spontaneous occurrence without a recognizable cause?

III. Timing of Events
- Onset of the clinical manifestation after exposure to the drug in question is an essential condition of causation.

IV. Drug level and evidence of overdose.
- The likelihood of a causal relationship is higher in situations where the dose consumed supports the diagnosis of an overdose.

V. Dechallenge
- Does the clinical manifestation resolve on drug withdrawal?

VI. Rechallenge
- Does the clinical manifestation return after re-exposure to the drug?

6.2.3 Application of this instrument to patients who believe they may have experienced digoxin toxicity is problematic because rechallenge at higher than normal doses would be inappropriate and many of the symptoms of digoxin toxicity are non-specific. Some of these non-specific symptoms, described above, include nausea, fatigue, loss of appetite, blurry vision. Non-specific symptoms, by definition, have plausible alternative etiologies. However, data on timing, drug level, and dechallenge are often available.

6.2.4 In the assessment of symptom causality for patients taking Digitek, the Kramer instrument may be helpful in estimating if the symptoms are plausibly related to digoxin. However, the instrument is not likely to assist substantially in determining if consumption of non-conforming tablets produced the symptoms. Among Digitek® users, findings of an elevated digoxin serum concentration and symptoms of digoxin toxicity may have resulted from over dosage using conforming tablets (e.g., taking extra pills, taking too high a prescribed dose, reduced clearance, etc). The finding of an increased digoxin serum level, therefore, does not establish that a patient consumed a non-conforming tablet. The FDA has indicating that Actavis detected 20 non-conforming Digitek® tablets among 4,800,000 inspected in one batch. Given this low observed proportion of non-conforming tablets, the probability of consuming a non-conforming tablet is very low. The probability of consuming two non-conforming tablets is

extremely low. As a result, the cause of digoxin toxicity in a patient who consumes Digitek® from a lot that includes both conforming and non-conforming tablets is more likely to be due to accidental overdose of conforming tablets or altered clearance of digoxin than exposure to non-conforming tablets.

6.3 Any estimation of the likelihood that a patient has experienced digitalis toxicity requires careful examination of clinical information.

6.3.1 Most of the manifestations of digitalis toxicity are non-specific and may have any number of other causes. Symptoms such as nausea and anorexia may be a result of other drugs, concurrent medical illness, stomach ulcer, etc. Signs such as ventricular ectopy or heart block may be the results of underlying heart disease, acute ischemia, or concurrent drug therapy. To estimate the likelihood that digoxin has caused a specific symptom or sign requires a careful review of the details of a patient's symptoms, the symptom course, concurrent drug therapy, the physical examination, diagnostic test results, serum drug concentrations, and subsequent clinical course.

6.3.2 To determine the correct oral dose for an individual patient (and, therefore, to determine how much digoxin is too much or too little for an individual patient), physicians must consider many pharmacokinetic factors (e.g., absorption, distribution, metabolism, excretion, dose duration), and factors that may affect dose response (e.g., age, dose interval, renal function, co-therapies, volume status, medication adherence, and social environment).

6.4 The nature and magnitude of digoxin's benefit varies among individuals.

6.4.1 Digoxin is used to treat different disease conditions among different individuals with differing medical histories. For patients with atrial fibrillation and no heart failure, for example, digoxin is indicated for rate control. The benefits of rate control may include improved exercise tolerance, reduction in palpitations, prevention of heart failure, prevention of hospitalization, elimination of need for other medications, etc. For any individual with atrial fibrillation and no heart failure, the profile of benefit may vary. For patients with systolic heart failure and no atrial fibrillation, the benefits may include improved exercise tolerance, reduced edema, prevention of hospitalization for CHF, etc. To assess the potential for on-going benefit of digoxin for an individual, clinicians would need to consider the specific circumstances of that individual.

6.5 There is high variability in the severity of digoxin toxicity.

6.5.1 Most instances of digitalis toxicity involve mild symptoms or signs that resolve with dose reduction or drug discontinuation. These include loss of appetite, nausea, apathy, fatigue, etc. However, some instances of digitalis toxicity involve disabling symptoms or life threatening events such as hyperkalemia, advanced heart block, or tachyarrhythmias. Accurate classification of severity for any single patient's experience of suspected digoxin toxicity requires careful examination of his or her clinical details.

6.6 Therapy for suspected digoxin therapy depends on the clinical manifestation, causation and severity.

6.6.1 Most instances of digitalis toxicity involve symptoms or signs that are not immediately life-threatening. These can be managed with general supportive measures (e.g., hydration, rest, and observation), dose adjustment or drug withdrawal, and evaluation for potential drug interactions. The pharmacologic half-life of digitalis is 1.5 to 2 days which means that the serum concentration can be expected to fall by 50% in that time.

6.6.2 Some manifestations of digitalis require more urgent or specific therapy. Use of digoxin-specific FAb antibody (Digibind®) may be indicated for severe hyperkalemia, ventricular arrhythmias, heart block or symptomatic bradycardia unresponsive to atropine, or hemodynamic instability. Hyperkalemia may also require therapies to help lower serum potassium and promote excretion of potassium.

6.7 One extra dose of digoxin is not likely to produce serious toxicity

6.7.1 In clinical medicine, it is not uncommon for patients to inadvertently take an extra dose of prescribed medication. An extra dose of a drug with a narrow therapeutic index, like digoxin, is more likely to cause harm than an extra dose of a drug with a broad therapeutic index. However, even for digoxin the risk for harm is very small.

All these opinions are expressed to a reasonable degree of medical certainty.

WALTER N. KERNAN, M.D.

Exhibit A

Curriculum Vitae
Walter N. Kernan, M.D.

CURRICULUM VITAE

Walter N. Kernan Jr., M.D.

Work Address: Department of Internal Medicine
Suite 515
2 Church Street South
New Haven, CT 06519
Telephone (203) 764-7000
Fax (203) 764-7000
Email: walter.kernan@yale.edu

EDUCATION

Dartmouth Medical School M.D.	1984
Harvard College B.A.	1978
Buxton School	1974

POSTGRADUATE TRAINING

1987-1989	Robert Wood Johnson Clinical Scholars Program, Yale University School of Medicine
1985-1987	Resident in Medicine, Johns Hopkins Hospital
1984-1985	Intern in Medicine, Johns Hopkins Hospital

FACULTY POSITIONS

2005-	Professor of Medicine, Yale University School of Medicine
1995-2005	Associate Professor of Medicine, Yale University School of Medicine
1990-1995	Assistant Professor of Medicine, Yale University School of Medicine
1989-1990	Instructor in Medicine, Yale University School of Medicine

CURRENT CLINICAL AND TEACHING RESPONSIBILITIES

Attending Physician, Yale-New Haven Hospital
Course Director, Ambulatory Component of the Internal

Medicine Clerkship
Director, General Internal Medicine Clinical Conference

BOARD CERTIFICATION

American Board of Internal Medicine 1987 (September)
National Board of Medical Examiners 1985

MEDICAL LICENSE

Connecticut 1988-present (license number 028405)

PROFESSIONAL ORGANIZATIONS

American College of Physicians (Fellow)
Society of General Internal Medicine
Sydenham Society
Clerkship Directors in Internal Medicine
Stroke Council, American Heart Association
Member, Society for Clinical Epidemiology and Health Care Research (SCHR)

HONORS

Master of Arts, privatim, Yale University, 2007
Society of Distinguished Teachers, Yale University School of Medicine 2005
Dean's Medical Education Farr Prize, Yale University School of Medicine 1997
The Medical Educator Award of the Society of General Internal Medicine (New England Region) for Distinction in Medical Education 1996
Julian & Melba Jarrett Memorial Award 1984
Upjohn Achievement Award 1984
Mosby Scholarship Book Award 1983
Alpha Omega Alpha, Dartmouth Medical School 1983
CIBA Award for Outstanding Community Service 1982

EDITORIAL BOARDS

Stroke (7/1/07 – 6/30/10)
Stroke (7/1/10 – present)

REVIEWER-ABSTRACTS

International Stroke Conference 2010
International Stroke Conference 2011

REVIEWER-JOURNALS

American Journal of Medicine
Annals of Internal Medicine (last 6/07)
Annals of Neurology (last 11/08)
Archives of Internal Medicine
Archives of Neurology (last 6/07)
BMC Medical Education
Cerebrovascular Diseases (last 10/09)
JAMA (last 2/10)
Journal of Clinical Endocrinology & Metabolism (last 7/10)
Journal of General Internal Medicine
Lancet (last 7/07)
Lancet Neurology (last 12/08)
Mayo Clinic Proceedings (last 1/05)
Nature Clinical Practice Neurology (last 6/3/07)
Neurology (last 5/10)
New England Journal of Medicine
Stroke (last 6/10)
Teaching and Learning in Medicine (last 7/07)

SERVICE TO ORGANIZATIONS

Organization	Role	Dates
American Heart Association/American Stroke Association	Member, Scientific Statements Oversight (SOC) Committee	2010-2012
American Heart Association	Member, Secondary Prevention Writing Group	2008-Present
Clerkship Directors in Internal Medicine	Member, Nominating Committee	2006
Clerkship Directors in Internal Medicine	Member, Task Force on Enhancing Student Interest in Internal Medicine Careers	2005-2006
Stroke Prevention in Small Subcortical Stroke (SPS3) Trial	Member, Events Adjudication Committee	2005-Present
Clerkship Directors in Internal Medicine	Chair, Nominating Committee	2005
Clerkship Directors in Internal Medicine	Past President	2005
The Stroke Association (British)	Grant Reviewer	2004
Clerkship Directors in Internal Medicine	President	2004
Alliance for Academic Internal Medicine	Member, Board of Directors	2003-2006
Clerkship Directors in Internal Medicine	President-Elect	2003
Clerkship Directors in Internal Medicine	Treasurer	2002
National Board of Medical Examiners	Member, USMLE Step 2 Test Material Development Committee for	2000-2002

	Ambulatory Medicine	
Clerkship Directors in Internal Medicine	Member, Council	1999-2002
National Board of Medical Examiners	USMLE Step 2 Test Material Development Committee for Medicine	1998-2000
Health Resources Service Administration	Member, Advisory Committee to the Bureau of Health Professions on Implementation of the Health Professions Education Partnership Act of 1998	1998
National Board of Medical Examiners	Member, USMLE Step 2 "R set" Task	1996

SERVICE TO THE NATIONAL INSTITUTES OF HEALTH

Committee	Role	Institute	Dates
Special Emphasis Panel ZNS1 SRB-M (74)	Member	NINDS	2010
NSD-C Initial Review Group	Member	NINDS	2010
NST-1 Review Committee	Standing Member	NINDS	2009-Present
Special Emphasis Panel/Scientific Review Group ZNS1 SRB-B (18)	Member	NINDS	2009-2010
Special Emphasis Panel ZNS1 SRB-R (43)	Member	NINDS	2009
Review Committee ZNS1 SRB G (41)	Member	NIH	2009
Special Emphasis Panel/Continuing Clinical Trials ZNS1 SRB-G (39)	Member	NINDS	2009
Special Emphasis Panel/ARRA Revisions ZNS1 SRB-G (38)	Member	NINDS	2009
Special Emphasis Panel ZNS1 SRB-G (37)	Chair	NINDS	2009
NST-1 Review Sub Committee	Ad-Hoc Member	NINDS	2009
Special Emphasis Panel/Scientific Review Group ZHL1 CSR-G (M1)	Member	NINDS	2009
Special Emphasis Panel/Scientific Review Group ZNS1 SRB-G (36)	Member	NINDS	2009
NSD-K Initial Review Group	Standing Member	NINDS	2008-2009
Special Emphasis Panel, Continuation CT Review# ZNS1 SRB-G (33)	Member	NINDS	2008
Special Emphasis Panel, CT # ZNS1 SRB-G (31)	Member	NINDS	2008
Special Emphasis Panel, SPOTRIAS Review # ZNS1 SRB-G (29)	Member	NINDS	2008

Special Emphasis Panel, Clinical Trial Review # ZNS1 SRB-B (11)	Member	NINDS	2008
Special Interest Panel, Clinical Trial Review # ZNS1 SRB-B (08)	Chair	NINDS	2008
Special Emphasis Panel, TRAINING # ZNS1 SRB-G (28)	Member	NINDS	2008
Special Emphasis Panel, Clinical Trial Review # ZNS1 SRB-B (09)	Member	NINDS	2008
Special Emphasis Panel, Continuing Clinical Trials Review # ZNS1 SRB-G (26)	Member	NINDS	2008
Special Emphasis Panel, SPOTRIAS Review # ZNS1 SRB-G (21)	Member	NINDS	2007
Special Emphasis Panel, NETT Review # ZNS1 SRB-G (23)	Member	NINDS	2007
Special Emphasis Panel, Continuing Clinical Trials Review # ZNS1 SRB-G (22)	Member	NINDS	2007
Special Emphasis Panel, NSDK Review # ZNS1 SRB-W (26)	Member	NINDS	2007
Special Emphasis Panel, WARCEF Review # ZNS1 SRB-K (46)	Member	NINDS	2006
Special Emphasis Panel, SPOTRIAS Review # ZNS1 SRB-G (41)	Member	NINDS	2006
Special Emphasis Panel, SPOTRIAS Review # ZNS1 SRB-M (30)	Member	NINDS	2006
NSD-K Initial Review Group	Standing Member	NINDS	2005-2008
Special Emphasis Panel, Acute Stroke Therapies Review # ZNS1 SRB-G (06)	Member	NINDS	2005-2006
Special Emphasis Panel, SPOTRIAS Review # ZNS1 SRB-M (30)	Member	NINDS	2005
Stroke Primary Prevention Task Force	Member	NINDS	2004
Support Systems for Providers Task Force	Member	NINDS	2002
Clinical Research Collaboration	Member	NINDS	2002
Primary and Secondary Stroke Prevention, Stroke Progress Review Group	Member	NINDS	2001
Health Services Implication, Stroke Progress Review Group	Member	NINDS	2001

COMMITTEE ASSIGNMENTS AT YALE SCHOOL OF MEDICINE

Committee	Role	Dates
YCCI/CTSA Career Oversight Committee	Member	2009
YCCI/CTSA Grant Review Committee	Member	2009
Search Committee for Chair of Neurology	Member	2008-2009
Ad Hoc Dean's Advisory Committee on Student Grievances	Chair	2008

Term Appointments & Promotions Committee	Member	2006-Present
Medical Library Committee	Member	2006-Present
Education Council, Department of Medicine	Member	2004-Present
Part-Time Faculty Appointments and Promotions Committee	Member	2000-Present
Physicians' Associate Program Review Committee	Member	1995-1996

INVITED TALKS OUTSIDE HOME INSTITUTION

Topic	Event	Institution/Location
2003		
Prevention of Stroke	Grand Rounds	Middlesex Hospital Middletown, CT
2004		
Secondary Prevention of Stroke	Grand Rounds	Waterbury Hospital Waterbury, CT
2005		
Dysglycemia and Stroke	Grand Rounds	Hartford Hospital Hartford, CT
Secondary Prevention of Ischemic Stroke	Grand Rounds	Saint Francis Hospital Hartford, CT
Teaching Quality of Care in the Medicine Clerkship	Forum	American Board of Internal Medicine Carmel, CA
Insulin Resistance Intervention after Stroke (IRIS) Trial	Staff Meeting	Hartford Hospital, Hartford, CT
Medical Complications of Stroke	Grand Rounds	St. Mary's Hospital Waterbury, CT
2007		
How to Manage Diabetes Mellitus for Stroke Prevention	International Stroke Conference	American Heart Association San Francisco, CA
The Hemorrhagic Stroke Project	Society Meeting	Boston Stroke Society Boston, MA
Advances in Stroke Prevention	Conference	Sunrise Hospital and Medical Center Las Vegas, NV
Insulin Resistance and Vascular Disease: From Pathophysiology to Therapy	Grand Rounds	The University of Arizona in Tucson Tucson, AZ
Insulin Resistance and Vascular Disease: From Pathophysiology to Therapy	Grand Rounds	New York University New York, NY
Confirmation of the Association between PPA and Risk for Hemorrhagic Stroke: The Story of the Hemorrhagic Stroke Project and its Legal Fallout	Seminar for Neurology Residents	University of Texas at San Antonio San Antonio, TX

Facilitating Student Learning	Symposium	AAMC Annual Meeting Washington, DC
Insulin Resistance and Risk for Stroke: Opportunities for Secondary Prevention	"Lunch & Learn"	NINDS, Clinical Research Collaboration Teleconference
Insulin Resistance and Risk for Stroke	Conference	Massachusetts General Hospital Boston, MA
IRIS: Treating Insulin Resistance to Prevent Stroke	Symposium	American Academy of Neurology Boston, MA
2008		
Glycemic Control for Preventing Stroke and Vascular Disease	International Stroke Conference	American Stroke Association, New Orleans, LA
Management of Metabolic Disease for Secondary Stroke Prevention	Symposium	Puerto Rican Academy of Neurology San Juan, Puerto Rico
Management of Metabolic Disease for Secondary Stroke Prevention	Grand Rounds	University of Western Ontario Ontario, Canada
Management of Metabolic Disease for Secondary Stroke Prevention	Grand Rounds	The Hospital of Central Connecticut New Britain, CT
Simulation: A Technique in Search of Meaning in the Medicine Clerkship	Lecture/Debate	CDIM Annual Meeting Orlando, FL
2009		
Metabolic Basis of Stroke Risk	Seminar	Newcastle University United Kingdom
Improving Insulin Sensitivity to Prevent Recurrent Stroke: The Insulin Resistance Intervention after Stroke (IRIS) Trial	"Lunch & Learn"	NINDS, Clinical Research Collaboration Teleconference
Stroke Risk Reduction in CT: An Opportunity for Primary Care Physicians	Conference	Connecticut Department of Public Health
Is There a Role for Tight Diabetes Control after Stroke?	Conference	American Heart Association Scientific Session 2009 Orlando, FL
2010		
Metabolic Syndrome and Stroke	Teleconference	Alberta Provincial Stroke Strategy Alberta, Canada
The Polypill: A Promising Strategy for Primary Prevention of Stroke	Conference	27th Princeton Conference on Cerebrovascular Disease Boston, MA

INVITED TALKS AT HOME INSTITUTION

Topic	Event	Institution
2006		
Silent Partners, Shattered Mission: The Value of Time, Observation and Communication between Teacher and Learner in Medical Education	Grand Rounds	Yale
2007		
Diabetes and Stroke	LM Brass Memorial Stroke Symposium	Yale
2008		
Efficient Assessment of Student Competence in the Office	Faculty Development Symposium	Yale
The History and Future of the Primary Care Crisis in the United States	National Primary Care Week	Yale
2009		
Measuring and Maintaining Drug Adherence in Long-Duration Studies: The IRIS Experience	General Internal Medicine Research in Progress	Yale
2010		
Hypertension Diagnosis and Management for Psychiatrists	Hypertension Grand Rounds	Yale Psychiatric Hospital

CURRENT RESEARCH

A randomized trial of the effectiveness of pioglitazone, compared with placebo, for prevention of ischemic stroke and MI among non-diabetic men and women with a recent transient ischemic attack or ischemic stroke and insulin resistance.

GRANT FUNDING

Title	Source	Dates	Support
CURRENT			
Insulin Resistance Intervention after Stroke (IRIS) Trial	NIH/NINDS	9/15/09-8/31/10	70%

Insulin Resistance Intervention after Stroke (IRIS) Trial	NIH/NINDS	7/1/04-7/30/09	70%
COMPLETED			
Temporal Trend in Insulin Resistance After Acute Stroke	Takeda Pharmaceuticals North America	12/17/01-3/18/03	10%
The Study of Insulin Resistance After Stroke-IIa	Takeda Pharmaceuticals North America	12/19/01-11/28/02	10%
The Study of Insulin Resistance After Stroke-II	Takeda Pharmaceuticals North America	1/1/01-11/28/02	10%
The Hemorrhagic Stroke Project	Non-Prescription Drug Manufacturers' Association	8/94-8/99	40%
The Women's Estrogen for Stroke Trial	NIH/NINDS	7/93-7/31/02	40 - 50%

PAPERS

1. Smith CR, Petty BG, Hendrix CW, Kernan WN, Garver PL, Fox K, Beamer A, Carbone CR, Threlkeld M, Leitman PS. Ceftriaxone compared with cefotaxime for serious bacterial infections. J Infect Dis 1989;160:442-447.

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Revised 7/1/10
Kernan\CV

Exhibit B

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Exhibit C

Walter N. Kernan, M.D.
Deposition and Court Testimonies in Past 4 Years

	My Client				Testimony	
Date of most recent Testimony	**Type***	**Name**	**State**	**Issue**	**Deposition**	**Court**
2/05	D	David Glabman	FL	SLE	Yes	Yes
8/05	D	Nature's Sunshine	PA	Stroke/Ephedra	Yes	No
11/05	D	Sho Me Natural	FL	Stroke/Ephedra	Yes	No
8/06	D	Phoenix Health	OK	Stroke/Ephedra	Yes	No
11/06	P	Andrea Daugherty	NJ	Cervical Cancer	Yes	No
1/07	D	Steven Urcioli	CT	Renal Cell Cancer	Yes	No
3/08	D	Gary Ferenchick	MI	Histoplasmosis	Yes	Yes
7/08	D	Miriam Hospital	RI	MI Prevention	Yes	Yes
8/08	D	The Pantry	FL	Stroke/Ephedra	Yes	No
10/08	D	David Salm	CT	MI Prevention	Yes	Yes
8/09	P	Sheila Matthews	FL	Epidural Abscess	Yes	Yes
8/09	D	Kelli Naylor	RI	MI diagnosis	Yes	No
9/09	D	Shahmaz Hussein	CT	GI bleeding	Yes	No
D=Defendant P=Plaintiff						